Modern Concepts of Cardiovascular Disease

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HEART FAILURE

PART I

This discussion will be limited largely to conditions in which an inability of the heart to perform a sufficient amount of work is the primary cause of disability or death. Every fatal disease ultimately involves heart failure, but in this review it will be impossible to consider conditions in which the cardiac disorder is not a primary factor in disability.

PHYSICAL PRINCIPLES

For either of two basic reasons the heart can fail to deliver to the arteries suitable amounts of blood under proper pressures. The heart may have either an inflow rate inadequate to fill it properly in diastole or it may not be able to do the work of expulsion. The first condition results whenever the ratio of blood volume to vascular bed volume decreases, either from loss of blood volume or from vasodilation.* The second situation occurs when the heart muscle is unable to perform the mechanical work imposed upon it.

The most fruitful way for the physiologist and the physician to look upon the heart is as a regulated pump-engine.[1] It is never appropriate to ignore the basic nature and function of a body organ in dealing with it from a clinical viewpoint. In the case of the heart this means that the physician dealing with cardiac problems should always orient his thinking with relation to its basic physiology. Pathologic changes in valves, excitation and conduction mechanism, coronary flow, the myocardium and the pericardium each produces its effects by altering the work capacity. Only in so far as the work capacity is or may be impaired is the pathologic process consequential to the heart itself. Vegetations may, of course, be significant as sources of emboli which may close blood vessels elsewhere.

Two factors determine the work output of any machine, (a) the manner in which it is loaded, and (b) the manner in which energy is supplied to it for performance of work. In the case of the heart the manner of loading depends largely upon two factors, (a) the filling pressure and (b) the arterial pressure.[2] These factors deserve further discussion.

The filling pressure for the heart ultimately depends upon the pressure in the great veins. The pressure in the ventricle at the end of its diastole is, of course, influenced by atrial contraction, but venous pressure determines atrial filling as well as ventricular filling before atrial systole, and therefore controls the ventricular filling due to both processes. The pressure in the great veins is, however, influenced by a variety of factors. The state of contraction of peripheral vessels, especially capillaries and venules, is of first importance. In the absence of heart failure either a dilation of the small veins or a decrease in blood volume causes a lowering of pressure in the great veins. In both of these situations the venous system is relatively too large for the available blood to fill it. But in addition the work capacity of the heart itself influences filling pressure. Whenever the heart becomes unable to eject in systole the volume of blood entering in diastole the residual blood in the heart increases.[3] Each such increment raises the early diastolic intracardiac pressure because the heart is an elastic reservoir. When an elastic bag is distended the pressure within it rises. If the elastic properties of the bag are simple the relation between pressure and volume can be calculated readily from the coefficients of elasticity. The elastic properties of heart muscle are not simple, however.

The heart muscle is a visco-elastic body and shows the phenomenon of hysteresis to a marked degree.[3] As a result the rise in intraventricular pressure occurring with chronic distention is much less than would be anticipated from its coefficient of elasticity before stretching. The phenomenon of hysteresis in heart muscle has been studied extensively. The elasticity of the myocardium falls after the heart has been made to work against heavy loads for some time. Working against high pressure the residual blood increases, as was noted above, and with this the decrease of elasticity occurring as a result of hysteresis is a useful adaptive process because it prevents the venous pressure from rising as high as it might otherwise do. Elevation of venous pressure beyond that necessary to fill the heart is disadvantageous, since it produces edema and other undesirable effects.[4]

The heart does work more economically against moderate arterial pressures and with high output than it does against high pressures and lower volume outputs.[5] Thus a hypertension is a disadvantage to the heart as a working machine, not simply because the cardiac work is increased by the elevated resistance, but also because the heart is less efficient with high pressure loads.

THE MECHANISM OF HEART FAILURE

From a fundamental viewpoint an engine, such as the heart, can "fail" to carry a load of work laid upon it for one or both of two reasons. It can so fail either because it cannot liberate energy or because it cannot effectively utilize the energy liberated, or a combination of both.[6] There are no other known possibilities for engine failure. The heart as a pump-engine fails for both of these reasons under various circumstances, but ordinarily mainly for the second, namely an inability to utilize liberated energy economically.[7] In other words, heart failure appears ordinarily to be an efficiency failure.[8,9,10,11] These assertions require amplification and supporting evidence.

*Inadequate filling also occurs when constrictive pericarditis is present. In this instance the inflow rate under diastole is reduced because of the mechanical impediment to stretching of heart muscle.

A very substantial literature exists bearing on experimental heart failure. In eight major pieces of investigation by separate authors all but two workers agree that spontaneous heart failure in experimental animals with isolated or intact hearts is primarily an efficiency failure. One worker has published the results of several experiments purporting to dispute this view.[13,15] In two experiments[12] this worker observed no change in efficiency with failure, but obtained a progressively declining coronary blood flow in those two instances. A declining coronary flow is not characteristic of failure in other instances. In these cases the myocardium in part or in whole had less oxygen delivered to it and the failure was probably due to that inadequacy. A third published experiment[13] showed an instance in which the oxygen content of the blood fell from 12 to 6 volumes per cent over the period of observation, while the calculated "efficiency" of liberated aerobic energy increased. It has been pointed out that when anaerobic processes occur calculations of "efficiency" from liberation of energy by oxidative processes are meaningless.[14] When a sizeable fraction of cardiac contraction energy comes from anaerobic processes the "efficiency" of oxidative work can and does exceed 100 per cent. Obviously calculations of "efficiency" from oxygen consumption and performed work have meaning only when all energy is derived from oxidation. This is the assumption implicit in all indirect calorimetry and cannot be ignored in studies of the metabolism of the heart. The second worker[15] whose reports cast doubt on the occurrence of failure of efficiency as the usual mode of spontaneous heart failure has employed a fallacious method of measuring oxygen consumption, as has previously been discussed at length.[16,17] The essence of the fallacy lies in the fact that coronary sinus blood is not a representative sample nor a constant fraction of total coronary blood flow,[18] as was assumed by the worker in question.

In a series of experiments numbering several hundred, in which the total oxygen consumption has been measured, it has been found that unless the coronary flow declines or oxygen tension in the blood falls, the process of failure is associated with a decline in cardiac efficiency as measured by indirect calorimetry in isolated hearts[14] or in the intact animal.[19] Consequently it may be stated with fairly great assurance that in the isolated heart, the heart lung preparation and in the intact animal spontaneous failure and failure induced by anesthetic agents are primarily failures of efficiency, so long as supply of oxygen is adequate for aerobic metabolism.

THE INFLUENCE OF VARIOUS FACTORS ON HEART FAILURE

The most striking reversal of the falling cardiac efficiency in failure is seen upon administration of the cardio-active glycosides.[10,20,21,22,23] Various glycosides of the digitalis type bring about large increase in the efficiency of the heart. This occurs independent of heart rate or of changes of the flow of blood in the coronary arteries. It is without doubt the basic reason for the effectiveness of such glycosides in the treatment of the decompensated or decompensating heart because no other effect is known which could favorably influence the performance of the heart in the absence of rate changes, and the regular heart is improved by digitalis in failure,[24] just as the irregular heart is. The intimate mechanism by which digitalis increases the efficiency of the failing heart is unknown. It may not have such an effect, at least for a long period, on the heart of the normal individual. However, this question has never been studied directly. The decreased minute output ordinarily seen in normal subjects after administration of digitalis does not prove an absence of an efficiency effect because the cardiac output is not limited or controlled by efficiency except in failure. In fact the decrease in the size of the heart[25] seen after administration of digitalis in normal subjects speaks for a probable increase of efficiency, because the shorter muscle fibers liberate less energy in contraction than do lengthened ones.[26]

The fact that digitalis allows the human heart when decompensated to do more work at a shorter initial fiber length (as measured by maximum diastolic volume) than it does without digitalis, just as is the case in the experimental animal, lends strong support to the view that experimental failure and clinical congestive failure are comparable entities. This point has been questioned by some writers. The complete similarity of reactions in experimental and clinical heart failure with respect to (a) cardiac dilation, (b) elevation of venous pressure, (c) response to digitalis administration, (d) reaction to decreased load, and (e) deleterious effects of increased work by elevated heart rate and elevated inflows and pressures, leads one to feel fairly confident that the two involve chiefly similar processes.

To be sure, human heart failure shows some effects due to its chronicity not seen in an acute experiment. For example, cardiac dilation is usually associated with prior or simultaneous muscular hypertrophy. The latter is a phenomenon requiring the long-time imposition of loads heavier than normal on a myocardial element and does not occur in a day. However, it does occur in experimental animals when cardiac loads are increased as they are increased in the presence of thyroid intoxication or excessive muscular work.[27] Further, it has been shown that there is an increase in what has been called the static blood pressure in patients dying in a state of cardiac decompensation.[28] This has been proposed as a reason for the elevated venous pressure in association with heart failure. However, it must be recalled that tissue edema occurs in heart failure and that the blood vessels may not be able to dilate fully after death from congestive failure because of the excess tissue tension. Such an occurrence would have nothing to do with the primary mechanism of heart failure or its symptoms.

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